A DISSERTATION ON

THE STUDY OF MIGRAINE HEADACHE SPECTRUM IN PATENTS TREATED IN THE TERTIARY CARE HOSPITAL

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CERTIFICATE

This is to certify that the dissertation titled “THE STUDY OF MIGRAINE HEADACHE SPECTRUM IN PATENTS TREATED IN THE TERTIARY CARE HOSPITAL” is a genuine work done by Dr.R.Samuel Appadurai Alexander for the partial fulfillment of requirements of D.M (neurology) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held in August 2014, under the table guidance and supervision of Prof.Dr.S.GOBINATHAN . M.D.D.M (neurology), Professor and Head, Department of neurology , Government Stanley Medical College and Hospital, Chennai.

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DECLARATION

I, Dr.R.Samuel Appadurai Alexander , Solemnly declare that the dissertation titled, “THE STUDY OF MIGRAINE HEAD ACHE SPECTRUM IN PATIENTS TREATED IN THE TERTIARY CARE HOSPITAL” is a bonafide work done by me during the period of March 2013 to February 2014 . at Government Stanley Medical College and Hospital, Chennai. Under the expert supervision of Prof.Dr.S.GOBINATHAN . M.D.D.M (neurology) , Professor and Head, Department of neurology , Government Stanley Medical College and Hospital, Chennai.

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INTRODUCTION

Headache is very common disorder of symptom complex with multifactorial origin. It has world vide prevalence of 57% in males and 67% with life time prevalence of %. There about 300 types of headache identified. Still they are not classified under a single. The international society has classified headache in the year. Now the third criteria of classification has also come.

Headache is a more common disease that it becomes a major cause for the patient to seek the advice of the physician and/or a neurologist. The causes of headache are numerous. Therefore a systematic approach is essential to find out the cause of every headache patient to solve this problem. Many headaches are externally not detectible and internally not measurable. This is also good field of research since it lacks sufficient facts.

The neuroanatomy of headache was thought to be the vault of the skull but now it is change to the brain which is the seat of all types of headache. Where ever be the original of pain in the head, it is generally headache.

In B.C. during SUMARIN and EGYPT civilization the headache is described in poems and papyri respectively. Headache was described in VEDIC period also. Hippocrates (BC 460-370) and his Greek followers had described migraine as flashes of light, moderate to severe pain in the head neck both temporal legions and eyes with vomiting.

Many other neurologists and scientist described that the cause of head ache is obscure but could be vascular, the pain and aura are from the cortex spreading either unilateral of bilateral to the part of the head up to the neck and association with unrelated sensory symptoms like nausea or vomiting. Most of the people indulged in the research of migraine are themselves suffering from migraine attacks.

The migraine headache prevalence is towards the increasing percentage possibly because of the influence of various environmental factors which worsen the internal mechanisms of headache. India has a migraines head ache of 52% as against the global prevalence of 12% of the general population.

According to International headache society classification the headaches are classified in to

1. Primary headache
2. Secondary headache
3. Unclassified headache

The primary headache is classified as follows.

1. Migraine headache
2. Tension type headache
3. Cluster headache
4. Other types of headache and crania facial pain

Being a post graduate student in Govt. Stanly Medical College Hospital which is a higher specialty Tertiary Care Hospital, Chennai I am

able to see a good number of headache cases both as outpatient and inpatient. As I am also a migraine it stimulated me to take up this topic as a dissertation.

Migraine headache is a moderate to severe headache which incapacitates the person and decreases the quality of life. So any small improvement due to treatment will help the patient to very great extent of help.

No investigation is contributory to the diagnosis of migraine. All the conventional and specific investigation is negative. Any positivity rules out migraine. So migraine is a diagnosis of clinical entity. The clinical symptoms and signs should fit in to the criteria of migraine. Otherwise the diagnosis is incorrect and patient is burdened with wrong treatment and side effects.

Finally, migraine is the more common disease than with think of, more severe than we expert of, not any contribution from any investigation than we expert of, not completely curable but only controllable and manageable.

AIM AND OBJECTIVES, OF THE STUDY

AIM AND OBJECTIVES

To study the following:

I Migraine headache type prevalence

II a. Migraine with aura

b. Migraine without aura

III Migraine demographic factors

IV Migraine headache characters

V Investigations

VI Treatment

VII Follow up

In the same time in Germany arteriole Venous shunts, from external jugular vein were thought of causing migraine and lead to the use of ergotamine in migraine since ergotamine is a vasoconstrictor to arteriole venous shunts, this may decrease the shunt and thereby deoxygenation to the brain.

After 1960

The ergotamine came to use in the practice of treating migraine. It is also used as a prophylactic agent for migraine in combination with phenobarbitone nd belladonna alkaloids. This drug was designated as “BELLARGAL”

Subsequently other drugs like Methysergide and Cyproheptadine came in to use. Later Methysergide was withdrawn due to its side effects.

After 1968, mono amine oxidize inhibitor, PHENELZINE 15 mg three times daily for 2 years was used for migraine head ache.

ERIK SKINHOJ (1973) demonstrated that the cerebral flow was decreasing during aura of the migraine.

In USA, Michael Welch's group demonstrated that there was dropping Magnesium ion during migraine. This drop stimulated N-Methyl D-aspartic acid receptors which led to the use of NMDA Receptor antagonists in the treatment of migraine.

There are about 43 head ache societies Head ache Medicine has become a sub specialty in USA.

The neurotransmitors serotonin and nor adrenalin were gained importance other than acetylcholine. Migraine research has lead to the invention of SUNCT and cluster head ache which are totally different from migraine.

Pain of head ache in General – Neurophysiology

The pain in different types of head ache in general can be of acute or sub acute onset and fast or slow progression.

It can start unilaterally and confined to it or spread to opposite side and become holocranial.

It can start bilaterally and became holocranial.

Its radiation to the neck has significance.

The head ache by and large is in trigeminal nerve territory whose anterior end includes temporal, region, orbital, frontal, nasal, nasopharyngeal, Buccal, alveolar and oral cavity.

Posteriorly it extends in to the middle cranial fossa and vertex.

It has connections with C2 branch like arterial anastomotic connections. It also has spinal root being shared by cervical root

So that pain of trigeminal origin spreads to posterior cranial fossa and to both sides of the neck.

So previously the head ache of migraine is thought of from the vault of the skull but now pain in the head means pain in trigeminal territory which extends from face to the neck externally and mouth, orbit all cranial fossae internally.

The middle meningeal arterial dilation also produces pain which is supplied by the trigeminal nerve.

Pathophysiology of Aura – Vascular Theory

Vasoconstriction and vasodilatation theory is being postulated for migrainous aura. Here vasoconstriction occurs first followed by vasodilatation extra cranial vasodilatation produces pain of the migraine but the vasoconstriction produces transient oligaemia. Cortical Hypoperfusion of the brain in the visual cortex produces aura. So in clinical setting the aura of migraine is followed by the pain. The hypo perfusion in occipital cortex proceeds anteriorly at the rate of 2 to 3 min/tm. The magnitude of hypoperfusion is 25% to 30% and proceeds in the front independent of arterial territories in a wave form along the convolution of cortex and does

not cross central or lateral sulcus but reaches the frontal lobe via the Insula.Where as the perfusion of sub cortical structures is normal. The hypoperfusion last for 4 to 6 hours and produce contra lateral neurological symptoms

Before causation of hypo perfusion mainly in the tempera parietal regions. Head ache begins when the hypoperfusion spreads to the frontal region.

The vasodilatation produces edema and focal tenderness. It is per se is not the cause for head ache as other alteration of cerebral flow occurs more than simple vasoconstriction and dilatation.

Genetic Basis of migraine

Episodic migraine is a component of MELAS Syndrome. Mitochondrial DNA transmitted from the mother is responsible for this head ache. So all children of MELAS mothers are affected by migraine.

Genetic susceptibility plays a role of 50% in migraine transmission and the remaining 50% is due to environmental factors. There is 4 times of risk of offsprings of the parents suffering from migraine. The chromosome 19 was identified to carry the gene responsible for familial migraine which constituted 55% of patients of migraine.

There is familial transmission of migraine with hemiplegia. This is caused by the mutation of CACNLIA4 gene on chromosome 19. The other mutation of same gene produces spinocerebellar ataxia of type 2 and 6.

Role of Sympathetic nervous system – empty neuron theory

A lot of changes in Autonomous nervous system occur before, during and after a migrainous attack. Monoamine oxidizes inhibitors and reuptake blockers minimize these symptoms.

Stress, sleeplessness, shift of hormones, and low blood sugar can cause depletion of catecholamine by releasing them from there stores. But nor adrenaline and sympatho-mimitics like isometheptene along with monoamine oxidize inhibitors and reuptake blockers counter act the actions.

Adequate concentrations of certain Neurotransmitters at post ganglionic sympathetic terminals are maintained by endogenous dopamine, prostaglandin and adenosine by inhibition of release of nor epinephrine. This is caused by Dopamine antagonist prostaglandin systhesis inhibitors and adenosine antagonist.

Head ache

The eyes, ears, nose, nasopharynx, sinuses, oral cavity, oropharyanx, teeth and gums are pain sensitive structures of the face. The pain of these areas is dull, aching and not sharply localised. Throbbing nature of pain indicates a vascular etiology. The index of pain is how much it incapacitates a patient rather than how a patient dramatizes it. Another index of severity is a pain either awakens a person from sleep or prevents the sleep of an awaken person. For any facial pain the quality, intensity,

Location, radiation, the mode of onset, peaking, indemnity curve, duration, associated factors, aggravating factors and relieving factors should be taken into account.

The pain sensitive structure of the cranium are all structures from the skin to periosteum of skull, major arteries, the durra, pie, arachnoids, small arteries like middle meningeal artery superficial temporal artery the sensory and mixed cranial nerves like optic, occulomotor, trigeminal, facial, gloss pharyngeal, vagus and upper three cervical nerves. The walls of the blood vessels are traversed by the nerve fibers and the dilatation of the blood produce pain. Pain from intra cranial portion of internal cerebral artery is felt in the eye balls and both orbit temporal regions, eye-brows and supra orbital regions. Similarly pain from the middle meningeal artery is felt in the back of the eye balls and temporal regions.

The Trigeminal nerve transmits the pain in the supra tutorial regions like orbit, forehead, nasal cavity, oral cavity, anterior and middle cranial fossae. The facial nerve transmits pain from nasal orbital region, ear and throat.

The ninth, tenth and upper cervical nerves carry the pain from infra tutorial regions, vertex and up to the posterior cranial fossa.

Mechanism of head ache

Dilatation of arteries irrespective of his position whether extra cranial or intra cranial will produce head ache. Cerebral vasodilatation is caused by alcohol consumption, Histamine infusion, nitrites and seizures. Fever with throbbing quality confers a vascular origin. Extreme rise of blood pressure, malignant hypertension, pheochromocytomas actual activity and patient on monoamine oxidize inhibitors will have throbbing head ache.

Head ache may be due to a variety of causes. It can be sudden as in glaucoma, purulent, sinusitis, sub arachnoid, Haemorrhage, bacterial and viral meningitis. Brain tumours produce sub acute and chronic headaches. Chronic and recurrent head ache are of neurologic in origin. Cluster headaches are unilateral and orbit frontal. They are nocturnal, present for few days with lacrimation, stuffy nose, Rhinorrhea, injected conjunctiva and ptosis

Tension headache are generalized, non throbbing, feeling of tightness, of variable intensity, present for days to week or months, triggered by fatigue and associate with depression, worry and anxiety

Temporal arthritis headaches are either unilateral or bilateral, throbbing with thickened arteries, intermittent to start with and becomes continuous, present for weeks to months, without any provoking factors, associated with loss of vision, Fever, weight loss, and Polymyalagia Rheumatica with good results for steroid therapy.

Other causes of crania facial pain includes ocular causes, meningeal irritation, post lumbar puncture and exceptional head ache.

Mechanism of migraine Head ache

The Trigeminal vascular system is stimulated and the impulses pass through laminate I and II of the nucleus caudal is of the decending tract and nucleous of trigeminal nerve and dorsal horn of C2 and C3. From here they travel via thalamus to posterior sensory cortex. There is also cutaneours allodynia which can be prevented by trip tans therapy. Apart from this, the substance P, Calcitonin gene related peptide and neurokinin A are secreted by trigeminal axonal nerve terminals mediate a nociocepive neurogenic

inflammatory process resulting in Central transmission of pain impulse. The neurogenic pain of migraine is neurogenic inflammation supported by

serotonin and platelet changes with mast cell degranulation, activation of local cellular immune response, leakage of plasma proteins from Dural vessels, vasodilatation and vascular endothelial activation usage of ergots, sumatriptants, valproicacid, highly selective serotonin receptor antagonist indomethacin for the treatment of migraine support this view of neurogenic implication.

There is also a neural mechanism behind migraine head ache the visual are activates cortical spreading depression which activates Trigeminal vascular afferents and stimulates a series of cortical meningeal and brainstem events which produce head ache. There is also parasympathetic stimulation and inflammation with up regulation of Nitric oxide syntheses by this the extra cephalic blood flow and neurogenic inflammation coupled to a cortical neuron electrical event.

Migraine Head ache

It is unilateral commonly and often pulsatile. Two clinical syndromes, migraines with aura and without aura

are identified both have a vague premonitory symptoms changes in mood and appetite.

Migraine with aura is associated with neurological symptoms before the onset of head ache an hour earlier. Usual symptoms are the commonest problems. Scientilating Scotomas, Hemianopia, Partial visual field loss flashes of lights, and colours zig-zag bright shining wavy line patterns. Illusion of distorted size and shape – Metamorphosia some time there may be migrainous aura without headaches It the pain occurs it is unilateral or bilateral with probing quality and moderate to severe, lasting for 4-72 hours.

Migraine without aura

It is an idiopathic recurring head ache lasting for 4-72 hours. Unilateral in location, with throbbing quality, Moderate to severe intensity, aggravated by physical activates and associated with nausea, photophobia and phonophobia.

Family history is present in 50% of the such patients may have premonitory symptoms 2-72 hou efore the onset of head ache

Migraine with aura is associated with Neurological Symptoms before the onset of headache. An hour earlier the visual symptoms occur. These are scintillating scotomas, hemianopia, partial visual filed loss, flashes of light and colors, and zig sag wavy pattern of lines occur. Metamorphsia also occurs. There may be aura only and not followed by the head ache. If the pain occurs it is unilateral or bilateral with throbbing quality which is moderate to severe, lasting for 4-72 hours.

Migraine without Aura

It is an idiopathic recurring head ache lasting for 4-72 hours, unilateral in location, with throbbing quality, moderate to severe in intensity,

aggravated physical activities and associated with nausea photophobia and phonon phobia. The aura is absent family history is present in 50% of such patients. Patients may have premonitory symptoms 2-72 hours before the onset of head ache which includes anorexia, hunger, drowsiness, tension and feeling of well being. The personality of migraines are usually ambitious perfectionists and intolerant.

The international classification of head ache disorders – II Edition

Part one: The primary Head aches

1. Migraine
2. Tension type of hea [16] e
3. Chester head ache and other trigeminal autonomic cephalalgia
4. Other primary headaches.

Part two: The secondary head aches

5. Head ache attributed to Head and/ or neck trauma
6. Head ache attributed to cranial or cervical vascular disorder
7. Head ache attributed to non vascular intracranial disorder
8. Head ache attributed to a substance or its withdrawal
9. Head ache contributed to infections
10. Head ache contributed to disorder of homoeostasis

11. Head ache or facial pain attributed to disorder of cranium, neck, eyes, ears, nose, sinuses, mouth, teeth or other of facial or cranial structures

12. Head ache attributed to psychiatric disorders

ICHD-2 criteria for migraine without aura

B. At least five attacks ful[17] the following criteria B to D

C. Head ache attacks lasting for 4-72 hours either untreated or unsuccessfully treated

D. Head ache has at least two of the following characteristics:

1. Unilateral locations
2. Pulsatile quality
3. Moderate or sever pain
4. Aggravation by or casing avoided for routing physical activity. (e.g. walking or climbing stairs)

E. During the Head ache attacks, at least one of the following

1. Nausea and / or vomiting
2. Photophobia and phonophobia

F. Symptoms not attributed to other disorder

Revised ICHD-2 criteria for migraine Head ache with typical aura

A. At least two attacks fulfilling the following criteria B to D

B. 1. Aura consisting of at least one of the following but no motor weakness.

2. Fully reversible visual symptoms including positive features (e.g. flickering lights, spots or lines) and /or ergative features (i.e. loss of vision)

3. Fully reversible sensory symptoms including positive features (i.e. pins and needles) and /or negative features (i.e. numbness)

4. Fully reversible dysphasic speech disturbance

C. At least two of the following

1. Homonymous visual symptoms and/or unilateral sensory symptoms.

2. At least one aura symptoms develop gradually over $\geq$ 5 minutes and/ or different aura symptoms occur in succession over $\geq$ 5 minutes.

3. Each symptom lasts $\geq$ 5 and $\leq$ 60 minutes

D. Head ache fulfilling criteria B to D in the criteria of migraine without aura. Head ache beings during the aura or follows the aura within 60 minutes.

E. Symptoms are not attributed to another disorder

Clinical features of migraine

Migraine without aura is the most frequent type of vascular head ache. According to the International Head Ache Society Criteria it has head ache which is moderate to severe, throbbing quality, unilateral lasting 4-72 hours aggravated by routine of stressful work, associated with nausea and/or vomiting, photophobia aı 19 onophobia with multiple episodes.

Migraine with aura has sensory, motor or visual symptoms that proceed the onset of head ache. It is associated with neurological disturbances during the head ache which fulfills the criteria of Migraine.

Migraine with aura and without head ache has only aura fulfilling the criteria. There is no head ache.

Complicated Migraine has been used for types of Migraine where a Migraine attack is left with a transient or a persisting neurological deficit.

Basilar Migraine is the term used if Migraine has brain stem dysfunction like vertigo, dysarthria blindness, diplopic, tinnitus and perioral paraesthesia lasting for 20-30 minutes and followed by occipital throbbing head ache. Though full recovery is the rule, the patient may have at least sensorium disturbance with confessional state for 5 days.

Biker Staff Migraine is one type of basilar Migraine running in families.

Ophthalmopelgic Migraine is rare form of recurrent attacks of unilateral orbital and retro orbital head ache lasting for 1 to 4 days. It is accompanied by ipsilateral ptosis. Paralysis of III in IV and VI Nerves

occasionally with vomiting. The neurological deficit remains for months together MRI shows thickening of III Nerve.

Retinal Migraine though it is rare, it has features of fully reversible scintillations, scotomas or blind 20 affecting one eye only with Migraine head ache either accompanying or following the other symptoms within 1 hour.

Familial hemiplegic Migraine: Migraine with aura associated with motor weakness usually hemi paresis lasting for 60 minutes to 24 hours with cerebella ataxia. Later in life similar motor weakness can occur with migraine without aura. There is a specific genetic pleomorphism is associated and chromosome 1, 2 and 19 are blamed.

Sporadic hemiplegic Migraine: If there is no family history of Migraine but aura, head ache and motor weakness still meeting the criteria then this term is used.

Migraine equivalents are neurological disturbances without aura and head ache. They are cyclical vomiting, nausea, anorexia with midline, paraumblical or poorly localised abdomen pain.

INVESTIGATIONS

- Visual evoked potential are of no value.
- There is an unclear relationship between the antiphospholipid antibody syndrome and Migraine

- Laser speckle contrast imaging the cortical spreading depression was shown to cause long lasting blood flow enhancement selectively with middle meningeal artery.
- CT Brain shows large areas of decreased attenuation over the hemisphere ipsilateral to the head ache especially if the head ache is severe and prolonged they are temporary and resolve in few days.

- In complicated Migraine the approximate areas of brain show infractions.
- MRI brain images show T2 signal within the sub cortical white matter.
- CT and MRI of brain are useful in exclusion the causes of recurrent head ache due to aneurysm
- EEG shows occipital spikes in 13-16 % of Migraine cases.

TREATMENT

1. NON PHARMACOLOGICAL

Patient is explained about Migraine that it is a medical problem only not psychological or cancerous. The normal CT and MRI brain reports would add to their reassurance. Explain them that even if Migraine is not curable it can be controlled and alleviated.

Simply relaxing in meaningless but avoiding stress and triggers are prudent. Smoking should be stopped. Alcohol and caffeine should be reduced medication is necessary which should be now and then reviewed and may be modified if necessary by the neurologist.

Sleep hygiene, avoiding excess diet and exercise programme are taught. Avoiding nitrite containingg foods like cold nuts and hot dogs are advised. Other food stuffs like red wine, chocolate chicken liver and pork are avoided. All canned and tinned foods containing monosodium glutamate are good for Migraine. Avoiding of strong odor perfumes strong smelling shampoos and soaps are avoided.

A psychologist help can be sought for the management of stress which is very difficult.

2. PHARMACOLOGICAL THERAPY

Symptomatic treatment like aspirin acetaminophen naproxen and ibuprofen may be started at the stage of aura itself because once vomiting and gastro intestinal symptoms develop no treatment is possible through mouth. Patient may be kept in a dark quiet room for rest. If patient gets sleep the head ache disappear after getting up.

Tripitans are new class of anti Migraine drugs which together with less selective ergot preparations are strongly agonist to 5 HT-IB receptor activities. Whose main action is cranial vasoconstriction and 5HT-ID reception activity whose action is cranial vasoconstriction and 5HT-ID receipts activity whose actions to release me sensory neuropeptides from perivascular trigeminal afferents.

Sumatriptan can be administered orally, intranasal and sub cutaneously. Orally it can be given in a dose of 25/50/100 mg every 2 hours to a maximum of 200 mg. Several trip tans like Almotriptan, elecriptan Nara triptan Rizatriptan and zolmitriptan are available. It is contra indicated in peripheral vascular disease, coronary artery disease and pregnancy. Narcoleptic agents and metaclopramide 10mg IV injections or chlorpromazine 5mg slow IV injections or repeated after 10 minutes to 30 minutes are effective.

3. ERGOTS have a role in symptomatic treatment of migraine they are both vasoconstrictors and vasodilators on dose based and resting tone of the target vessels. Parenteral and rectal perpetrations are more effective than oral preparations. They also act of 5HT receptors. 2 mg of ergot oral preparation is combined with an analgesic and caffeine combination. This is repeated every hour.

PROPHYLACTIC TREATMENT is carried out on patient to who have frequent and prolonged attacks. There is 50% reduction in half of this patient. Propranolol in dose of 80 to 240 mg per day given for 2-3 months orally. Any other β blocker can also be used. They should not be used for a long time for fear of their cardiac side effects.

The tricyclic antidepressants mainly with amitriptyline are used with unknown mechanism and good results are obtained. It is given in the dose of 10-150mg at night. Selective serotonin reuptake inhibitors and monoamine oxidize inhibitors are also useful. Calcium channel blockers like verapamil and flunarizine are used with unknown mechanism. Anticonvulsants like gabapentin 900 to 2400mg / day topiramate 75-200 mg per day can be used. Cyproheptadine and methysergide which is peripheral serotonin antagonist prevent platelet aggregation and weak antibody kimi activity is also present.

METHODOLOGY

In this hospital, 400 headache patients were selected from the department of neurology. 100 migraine patients were identified among them in a span of one year. They were further investigated, studied and followed up in detail from March 2013 to February 2014 for a period of one year at Govt. Stanley Medical College Hospital which is a tertiary care center.

The patients who were treated know Tamil well so that exact history could be obtained.

Using I.H.C.D. Criteria may were diagnosed as migraine belonging to various categories.

A proforma with diagnosis is prepared with usual analogue scales. MAIDAS schooling system was used in migraine headache patients.

Statistical analysis of migraine patients and disease with help of statistician was done and results and reports were displayed in bar chart and pie chart.

DISCUSSION SUMMARY AND CONCLUSION

The migraine headache spectrum start as a mild head discomfort on one end to neurological deficit to the other end through severe headache, vomiting, photophobia and phonophobia. This differs from one patient to other patients. They have to seek for a dark, quiet room and take rest until me headache is over which will take between 4-72 hours.

Migraine is externally not detected and internally not measurable. No investigation the reveals any abnormalities. Everyone would have suffered once in their life.

Migraine is a recurrent attack of headache and patient in frustrated. A little one in of great help for any migraine patients which will improve the quality of life.

The treatment of migraine is a multi disciplinary approach which needed Psychologist counseling in managing stress. Avoiding of certain dietary foods and certain habits which are triggers for migraine. Prophylactic and symptomatic, treatment plays a magic role in aborting a migraines episode.

Institution of one or two drugs among the array of antimigrainous drugs which suit the patient will reduce the agony of the episode.

In conclusion, though migraine in not curable, it is yet manageable and controllable. It can be aborted and episodes can be reduced. Patient care learns to live with migraine without much disturbance in quality of life. Reassurance always helps the patient and finally treating successfully a migraine gratifies the neurologists also.

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